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O'Melveny & Myers
610 Newport Center Drive
17th Floor
Newport Beach, CA 92660-6429

EXAMINER	
POHNERT, STEVEN C	
ART UNIT	PAPER NUMBER
1634	

NOTIFICATION DATE	DELIVERY MODE
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

Office Action Summary	Application No.	Applicant(s)	
	10/581,936	KAPPEL, ANDREAS	
	Examiner	Art Unit	
	Steven C. Pohnert	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *07 June 2006*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *11-29* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *11-29* is/are rejected.

7)☒ Claim(s) *11-21* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☒ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *6/7/2006*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Claim status

Claims 1-10 have been canceled.

Claims 11-29 are pending.

Claim Objections

1. Claims 11-21 are objected to because of the following informalities:

Claims 11-19 are objected to as they recite, “at least one of the fist” in the last step. This appears to be a typographical error and should be amended to “at least one of the first.”

Claim 18 is objected to under 37 CFR 1.75(c), as being of improper dependent form for failing to further limit the subject matter of a previous claim. Applicant is required to cancel the claim(s), or amend the claim(s) to place the claim(s) in proper dependent form, or rewrite the claim(s) in independent form. Claim 18 recites " GGG, GGA, GGC, and GGT” however the claim depends from claim 17, which requires the mutations, encode glutamic or aspartic acid. The DNA translation chart clears teaches that GGT, GGA, GGC and GGT are codons for glycine, while GAT, GAC, AAT and AAC are codons for aspartic acid (WWW.uq.edu.au/vdu/DNAtranslation.htm, 7/17/2007). Thus claim 18 contains subject matter that is broader in scope than claim 17.

Claims 19-21 recite, “at codon 599 of exon 15.” The recitation of "at codon 599 of exon 15” suggests there are at least 599 codons in exon 15. This objection can easily be overcome by amending the claim to recite “at codon 599 which is in exon 15.” Appropriate correction is required.

Specification

2. The disclosure is objected to because of the following informalities:

The specification recites "codon GGG, GGA, GGC, and GGT for aspartic acid" on page 3, 3rd line of 2nd paragraph. The DNA translation chart clears teaches that GGT, GGA, GGC and GGT are codons for glycine, while GAT, GAC, AAT and AAC are codons for aspartic acid (WWW.uq.edu.au/vdu/DNAtranslation.htm, 7/17/2007).

The specification on page 5, the first paragraph after the heading examples recites," ,,primary tumors" or ,, metastasizing tumors"" this appears to be a typographical error and should recite, " "primary tumors" or "metastasizing tumors."""

The specification on page 6, the last paragraph after the heading examples recites," ,,wildtype" or ,, mutant"" this appears to be a typographical error and should recite, " "wildtype" or "mutant."""

Appropriate correction is required.

Claim Rejections - 35 USC § 112

3. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 11-29 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

There are many factors to be considered when determining whether there is sufficient evidence to support that a disclosure does not satisfy the enablement requirement and whether any necessary experimentation is undue. These factors have been described by the court in re Wands, 8 USPQ2d 1400 (CA FC 1988). Wands states at page 1404,

"Factors to be considered in determining whether a disclosure would require undue experimentation have been summarized by the board in the Ex parte Forman. They include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims."

The nature of the invention and the breadth of the claims:

The claims are broadly drawn to a method of detecting malignancy of melanoma in cells in "any" patient by detecting the wildtype allele for codon 599 of the BRAF gene or "any" mutant allele of the BRAF gene.

The recitation of "patient" broadly encompasses any patient form any species including humans, dogs, cats, mice, whales, fruitflies, etc.

The claims are broadly drawn to the detection of malignancy in "any" patient sample. Any patient sample broadly encompasses melanoma sample, blood, urine, hair, buccal swab, etc.

The recitation of "a mutant BRAF sequence containing a mutation at codon 599" broadly encompasses any mutation at codon 599 as well as any other mutation in BRAF.

Claim 16 draws the claims to potential codons for valine at position 599 of the human BRAF gene.

Claims 17 draws the claims to the mutation selected from glutamic acid or aspartic acid.

Claim 18 draws the claims to codons of GAG (glu), GAA (glu), GGG (gly), GGA (gly), GGC(gly), and GGT(gly).

Claim 22 is drawn to method of detecting malignancy of melanoma by detecting hybridizing an oligonucleotide probe of exon 15 or a part of exon 15 comprising codon 599 and "counterstrands" with both a wildtype reporter comprising a sequence according to SEQ ID NO 5 and a first label and a mutant reporter selected from the group consisting of a sequence according to Seq. ID No. 6, a sequence complementary to Seq. ID No. 5, a sequence complementary to Seq. ID No. 6, a sequence with a homology of over 80% to Seq. ID No. 6, a sequence with a homology of over 80% to a sequence complementary to Seq. ID No. 5, and a sequence with a homology of over 80% to a sequence complementary to Seq. ID No. 6; and detecting the first label and second label. Thus the claim encompass a method of detecting melanoma without requiring the step of using DNA from a subject or patient, by forming a triple helix structure of a probe, wildtype reporter, and the mutant reporter.

Claim 22 appears to suggest the ability to detect a mutation by comparing a wildtype reporter of SEQ ID No 5 and a mutant reporter of SEQ ID NO 5.

Further the claim is drawn is drawn to "a sequence" or "a complement" or a sequence with 80% homology. The recitation of "a sequence" or "a complement" broadly requires any nucleic acid comprising a single nucleotide complementary to the SEQ ID NO. Further the recitation of 80% homology does not require the presence of codon 599 to be valine or gluatmic acid, but could be any nucleic acid triplet including a stop codon.

Claim 25 draws the art of exon 15 of claim 22, to SEQ ID No, complements of SEQ ID No 1, a part of SEQ ID No 1, comprising codon 599 or "any" allelic variant of SEQ ID No 1. "Any" allelic variant of SEQ ID No 1, could arguable be any nucleic acid sequence.

Claims 27 draws the oligonucleotide probe to "any" mutation of codon 599.

The amount of direction or guidance and the Presence and absence of working examples.

The specification teaches that a mutation in exon 15 of human BRAF had previous been described (V599E) (see page 1, 5ht paragraph).

The specification teaches allelic variants have sequence homology of more than 60% (page 3, paragraph 1).

The specification teaches that SEQ ID NO 5 is a wildtype reporter and SEQ ID NO 6 is a mutant reporter (page4, paragraph 2).

The specification teaches the preferred method is parallel hybridization of a labeled wildtype reporter and a mutant reporter under stringent conditions.

The specification suggests stringent conditions encompasses 60°C, 0.1 x SSC and 0.1% SDS (page 4, last paragraph). However, the specification does not define stringent conditions in this manner.

The specification teaches detection of the V599E mutation by PCR amplification and hybridization using a wildtype probe (SEQ ID No 5) and a mutant probe (SEQ ID No6) (page 5, last 2 paragraphs) in melanoma samples.

The specification provides no data or suggestion that the presence of the V599E mutation could be found in any other patient sample, or if found in other tissues the mutation would predictably be associated with malignant melanoma.

The specification teaches, “Surprisingly a correlation between the genotype and the malignancy of the tumor was found. The ,,wildtype" group (wt/wt) suffered from non-metastasizing melanoma (examples 1 to 12), whereas the ,mutant" group (V599E/wt; V599EN599E) suffered from metastasizing tumors (examples 13 to 29)” (se page 6, last paragraph). However examination of the data in table 1 demonstrates, while the non-metastasizing samples 1-12 were homozygous wildtype for the mutation, 6 of the 17 metastasizing tumors were also homozygous for the wildtype allele. Further example 15 did not provide a result so 8 of the 17 samples metastasizing tumors were not positive for the mutation. Only 3 of the 17 metastasizing tumors were homozygous for the mutations.

Thus the specification does not teach that the presence of the mutation is predictably associated with metastasizing tumors as 47% of the metastasizing tumors were not positive for the mutation. Thus it would be unpredictable to associate metastasis or melanoma with the presence of the mutation.

The specification does not teach the use of reporters with 60% or 80% homology to SEQ ID NO 1, SEQ ID No 5 or SEQ ID NO 6. The specification does not provide support for the predictable use of "any" complement, "any" fragment of the recited SEQ ID NO. Further the specification provides no support that a mutation of valine 599 to either glycine or aspartic acid occurs or is indicative of melanoma, malignant melanoma, etc.

The state of prior art and the predictability or unpredictability of the art:

Dong et al (Cancer Research (July 15, 2003)volume 63, pages 3883-3885) teaches the missense mutation V599E of the human BRAF gene was found in 17/24 (71%) samples with melanocytic nevi (benign skin condition)(table 1). Dong further teaches 13 of 21 patients (62%) with invasive melanoma or metastatic melanoma had the V599E mutation. Dong thus suggests the presence of the V599E mutation is not predictably able to identify melanoma or metastastic melanoma as it is found in other skin condition with a greater frequency.

Further, Uribe et al (American Journal of Dermatopathology(2003) volume 25, pages 365-370) teaches, "Our findings of a high frequency of BRAF mutations at codon 599 in benign melanocytic lesions of the skin indicate that this mutation is not sufficient by itself for malignant transformation" (abstract). Uribe teaches, "The major finding of

our study was the high frequency of the BRAF mutation at codon 599 in a series of benign (72%) and atypical (52%) melanocytic lesions of the skin, indicating the BRAF mutation at codon 599 should not be considered a feature of melanocyte malignant transformation" (see page 369, 1st column, 1st paragraph). Thus the combination of Uribe and Dong suggest the V599E mutation is not predictably associated with malignant melanoma.

Brenner et al (Trends in Genetics (2001) volume 17, pages 414-418) teaches that, "Here, the 'homology-implies-equivalency' assumption is restricted to a subset of homologs that diverged in the most-recent common ancestor of the species sharing the homologs. This strategy is useful, of course. But it is likely to be far less general than is widely thought. Two species living in the same space, almost by axiom, cannot have identical strategies for survival. This, in turn, implies that two orthologous proteins might not contribute to fitness in exactly the same way in two species" (see page 414, 3rd column last full paragraph). Brenner specifically describes that although the leptin gene homologs have been found in mice and humans, their affect is different (see page 414, 3rd column last paragraph-3rd column page 415). Brenner specifically teaches that the leptin gene in mice plays a major role in obesity, but no such effect has been demonstrated in humans due perhaps to the different evolutionary forces. Brenner thus teaches that the activity and function of genes in different species is unpredictable. Brenner thus teaches is would be unpredictable to associate a mutation in one species with a disease in another species, as the homologs often different functions.

The art of record is silent on a valine to glycine or valine to aspartic acid mutation at position 599 of human BRAF.

The level of skill in the art:

The level of skill in the art is deemed to be high.

Quantity of experimentation necessary:

In order to practice the invention as claimed one of ordinary skill in the art would first have to determine if the presence of the V599E mutation in human BRAF is predictably associated with detection of malignancy of melanoma cell. This would be unpredictable as the instant specification teaches 53% of metastatic tumors did not have the mutation, further Dong et al teaches that 71% of melanocytic nevi (benign skin condition) had the mutation. Dong further taught that 62% with invasive melanoma or metastatic melanoma had the allele. Finally, Uribe teaches, "The major finding of our study was the highfrequency of the BRAF mutation at codon 599 in a series of benign (72%) and atypical (52%) melanocytic lesions of the skin, indicating the BRAF mutation at codon 599 should not be considered a feature of melanocyte malignant transformation" (see page 369, 1st column, 1st paragraph). Thus the claims are not enabled for a method of detecting of malignancy in melanoma cells in as the specification and art teach the mutation is found in cells that are not malignant melanoma.

Further as the claims are not limited to humans the skilled artisan would have to determine if a mutation at position 599 of RAF is predictably associated with melanoma, malignant melanoma, etc in any other species. This would be unpredictable as Brenner

teaches that genes in different species often have different functions and thus do not display the same phenotypes due to the selective pressure on the species.

The specification provides no data or suggestion that the presence of the V599E mutation could be found in any other patient sample, or if found in other tissues the mutation would predictably be associated with malignant melanoma.

Further the specification and art provide not evidence to suggest that any mutation at position 599 of BRAF is indicative of something. The specification and prior art teach that the valine at position 599 has been found to be mutated to glutamic acid, however the specification and art provide not evidence that codon 599 is mutated to an aspartic acid or glycine as claimed. Further the specification and art do not teach any mutations at position 599 of human BRAF gene other than the V599E mutation.

The specification and art of record do not suggest that a method of hybridizing an oligonucleotide probe of exon 15 with wildtype reporter (SEQ ID NO 5) and mutant reporter (SEQ ID No 5 or SEQ ID No 6) would result in detection of a mutation. The claim as written appears to require the formation of a triple helix structure and it is unclear how this would allow discrimination of alleles. Further the claim is drawn to the use of SEQ ID No 5 as both the wildtype reporter and mutant reporter. It would thus be unpredictable to do a hybridization assay using the same probe to detect wildtype and mutants.

Further it would be unpredictable to detect a mutation at codon 599 of human BRAF using any allelic reporter or reporters that are 80% homologous to SEQ ID No 5 or SEQ ID NO 6. It would be unpredictable because there is nothing of record

suggesting the claimed reporters with 80% homology to SEQ ID NO 5 or SEQ ID NO 6 could discriminate a mutation at codon 599 of human BRAF and further the reporters as claimed could encompass any codon at position 599 including stop codons, methionine, etc and there is not evidence any amino acid other than valine or glutamic acid occurs at codon 599 BRAF in any species.

Therefor, in light of the breadth of the claims, the lack of guidance in the specification, the high level of unpredictability in the associated technology, the nature of the invention, the negative teachings in the art, and the quantity of unpredictable experimentation necessary to practice the claimed invention, it would require undue experimentation to practice the invention as claimed.

4. The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

5. Claims 11-29 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 11- 18 are indefinite because it lacks a positive active step relating back to the preamble. The preamble recites a method of a method of detecting malignancy of melanoma cells in a patient, however the last positive active step is drawn to detecting at least one of the first and second oligonucleotides hybridized to the patient sample. Therefore it is unclear as to whether the method is drawn to a method of detecting malignancy of melanoma cells in a patient or detecting at least one of the first and second oligonucleotides hybridized to the patient sample.

Claims 19- 21 are indefinite because it lacks a positive active step relating back to the preamble. The preamble recites a method of a method of detecting malignancy of melanoma cells in a patient, however the last positive active step is drawn to determining the presence of a mutation at codon 599 of exon 15. Therefore it is unclear as to whether the method is drawn to a method of determining method of detecting malignancy of melanoma cells in a patient or the presence of a mutation at codon 599 of exon 15.

Claims 22-29 are indefinite because it lacks a positive active step relating back to the preamble. The preamble recites a method of a method of detecting malignancy of melanoma cells in a patient, however the last positive active step is drawn to detecting at least one of the first and second oligonucleotides hybridized to the patient sample. Therefore it is unclear as to whether the method is drawn to a method of detecting malignancy of melanoma cells in a patient or detecting at least one of the first and second oligonucleotides hybridized to the patient sample.

Claim Rejections - 35 USC § 102

6. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

7. Claims 19-21 are rejected under 35 U.S.C. 102(b) as being anticipated by Dong et al (Cancer Research (July 15, 2003)volume 63, pages 3883-3885).

This rejection is presented as anticipating the active steps of the claimed invention. It is consistent with the enablement rejection as the claims do not require an active step of correlating the mutation with melanoma or malignant melanoma.

As noted in the MPEP 2111.02, "If the body of a claim fully and intrinsically sets forth all of the limitations of the claimed invention, and the preamble merely states, for example, the purpose or intended use of the invention, rather than any distinct definition of any of the claimed invention's limitations, then the preamble is not considered a limitation and is of no significance to claim construction." Accordingly, the claim language of " detecting malignancy of melanoma cells in a patient" merely sets forth the intended use or purpose of the claimed methods, but does not limit the scope of the claims.

Dong teaches PCR amplification and sequencing of human BRAF exon 15 and determining the presence of a glutamic acid at codon 599 (abstract and page 3883, 2nd column, last paragraph). Thus Dong teaches providing a patient sample, amplifying a patient sample, sequencing a patient sample and determining the presence of the codon 599 mutation is a glutamic acid.

Claim Rejections - 35 USC § 103

8. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

9. Claims 11-18, 22-29 are rejected under 35 U.S.C. 103(a) as being unpatentable over Dong et al (Cancer Research (July 15, 2003)volume 63, pages 3883-3885) as evidenced by NM_004333 GI:4757867 (published April 7, 2003, pages 1-4) in view of Gilles et al (Nature Biotechnology (1999) volume 17, pages 365-370).

This rejection is presented as anticipating the active steps of the claimed invention. It is consistent with the enablement rejection as the claims do not require an active step of correlating the mutation with malignant melanoma.

As noted in the MPEP 2111.02, "If the body of a claim fully and intrinsically sets forth all of the limitations of the claimed invention, and the preamble merely states, for example, the purpose or intended use of the invention, rather than any distinct definition of any of the claimed invention's limitations, then the preamble is not considered a limitation and is of no significance to claim construction." Accordingly, the claim language of "detecting malignancy of melanoma cells in a patient" merely sets forth the intended use or purpose of the claimed methods, but does not limit the scope of the claims.

Dong teaches PCR amplification and sequencing of human BRAF exon 15 and determining the presence of a glutamic acid at codon 599 (abstract and page 3883, 2nd column, last paragraph). Dong et al teaches a wildtype codon 599 of GTG and a mutant of GAG (see figure 1B). Dong et al teaches GenBank accession for BRAF is NM_004333 (see page 3833, 2nd column, last paragraph).

Dong et al does not teaches an assay providing a wildtype probe with a first label and a mutant probe with a second label. Dong does not teach detecting the hybridization of the first and second probes to the patient sample.

However, Gilles et al teaches a method of discriminating single nucleotide polymorphism by use of wildtype and mutant probes (see abstract). Gilles et al teaches his method allows rapid screening and the flexibility to customize chips (see page 365, 1st column, 2nd paragraph). Gilles teaches wildtype and SNPs allele sequences were differentially labeled (see page 365, 2nd column, last paragraph). Giles teaches the differentially labeled probes were scanned to detect the labels of the wildtype and mutant probes (see figure 2). Giles teaches his method allows discrimination of 4 separate alleles (see page 369, 1st column, last paragraph, to top of 2nd column).

Designing probes, which are equivalents to those taught in the art is routine experimentation. The prior art teaches the probes can be designed to wildtype and mutant sequences (see Gilles). Moreover there are many internet web sites that provide free downloadable software to aid in the selection of probes drawn from genetic data recorded in a spreadsheet. The prior art is replete with guidance and information necessary to permit the ordinary artisan in the field of nucleic acid detection to design probes. As discussed above, the ordinary artisan would be motivated to have designed and tested new probes to obtain additional oligonucleotides that function to detect mutations in the 599 codon of human BRAF and identify oligonucleotides with improved properties. Thus, for the reasons provided above, the ordinary artisan would have designed additional probes using the teachings in the art at the time the invention was

made. The claimed SEQ ID NOs are obvious over the cited prior art, absent secondary considerations.

Therefore it would have been prima facie obvious to one of ordinary skill in the art at time the invention was made to combine the V599E mutation of the human BRAF gene taught and the GenBank accession for BRAF is NM_004333 by Dong with the method of detecting mutations taught by Gilles using differentially labeled wildtype and mutant probes. The artisan would be motivated to combine the teachings of Dong, NM_004333 and Gilles because Gilles teaches his method allows for rapid discrimination of 4 separate alleles. The combined teachings of Dong, NM_004333 and Gilles would result in probes that are the same or functional equivalents of SEQ ID NO 1, SEQ ID No 5, and SEQ ID NO 6. The combined teachings of Dong, NM_004333 and Gilles would result in a more rapid method of detecting mutations in the human BRAF gene. The artisan would have a reasonable expectation of success as Dong and Gilles are both teach methods of detecting mutations and thus the artisan would merely be substituting one known method of mutation detection with another.

Summary

No claims are allowed.

Conclusions

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Steven C. Pohnert whose telephone number is 571-272-3803. The examiner can normally be reached on Monday-Friday 6:30-4:00, every second Friday off.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on 571-272-0735. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Steven Pohnert

/Sarae Bausch/
Primary Examiner, Art Unit 1634